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Case Report

Anaesthesia Section

Intraoperative Hyperkalemia Leading to Ventricular Tachycardia during Laparoscopic Renal Transplant

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ABSTRACT

Chronic kidney disease, consequent upon a variety of causes is a leading cause of morbidity and mortality. Renal transplantation is the preferred therapeutic approach for these patients. Hyperkalemia is a common complication observed intraoperatively during transplant surgery. Some of the causes of the same could be administration of succinylcholine, blood transfusion, metabolic acidosis and reperfusion injury.

We wish to highlight a case where during laparoscopic kidney transplant we encountered respiratory acidosis as one of the causes of hyperkalemia leading to ventricular tachycardia. In future, laparoscopic kidney transplant will become the standard of care similar to how laparoscopic donor nephrectomy is. It is thus, imperative for anaesthetists' to be well aware of challenges faced during this surgery, in order to ensure effective perioperative management.

Keywords: Anaesthesia, Complication, Respiratory acidosis

CASE REPORT

A 30-year-old male patient, diagnosed to have End Stage Renal Disease (ESRD) secondary to polycystic kidney disease, was scheduled for laparoscopic renal transplant. He was on maintenance haemodialysis for the same since two years. In addition, he received medications like nifedipine, clonidine and atenolol to aid in blood pressure control. The patient's most recent dialysis had been performed 12 hours prior to the surgery.

The preoperative general physical and systemic examinations were normal. Laboratory values before surgery were suggestive of haemoglobin of 10.8 g%, creatinine of 5.5 mg%, random blood sugar of 120 mg/dl, serum sodium of 144 mmol/L and serum potassium of 4.5 meq/L. A blood gas sample revealed a pH of 7.39, pCO_2 of 37 mm Hg, HCO^-_3 of 25 mmol/L and saO_2. ECG was suggestive of a normal sinus rhythm with normal QRS complex and T waves. Echocardiography showed a good left ventricular function (EF-60%) with mild left ventricular hypertrophy and no regional wall motion abnormality.

In the operation theatre, prior to induction, his baseline pulse was 98/min and blood pressure was recorded at 150/90 mm Hg. Pre-medications in the form of injection ranitidine 50 mg, ondansetron 4 mg, glycopyrrolate 0.2 mg and fentanyl 5 µgm/kg were administered intravenously. After pre-oxygenation and rapid sequence induction of anaesthesia with thiopentone sodium 7mg /kg and succinylcholine 1.5mg/kg, trachea was intubated with an 8.5 mm cuffed portex endotracheal tube. Anaesthesia was maintained with a mixture of oxygen, air, inhalational agent isoflurane and atracurium infusion (0.5 mg/kg/hr). Monitoring includes non invasive blood pressure, pulse oximetry, electrocardiogram, central venous pressure, capnography and peripheral nerve stimulator. Ventilation was controlled to keep $EtCO_2$ between 30-40 mmHg and peak airway pressure less than 35 cm of H_2O.

After induction, carboperitoneum was generated. The pulse rate increased from 98/min to 110/min. The surgery proceeded uneventfully for the next five hours. Two litres of normal saline was infused intraoperatively and the CVP was maintained around 16-18 mm Hg. A 500 mg of injection methylprednisolone, 100 mg furosemide and 100 ml of 20% mannitol were administered before the release of vascular clamps. Vascular anastomosis time was 48 minutes and urine output was established immediately on declamping. The blood pressure was around 160/90 mm Hg throughout the surgery but the heart rate gradually increased from 110 to 130/min after clamp release. ECG was normal except for sinus tachycardia.

At the conclusion of surgery, just before extubation the heart rate suddenly increased from 130/min to 170/min within a minute. Within seconds the rhythm changed to ventricular tachycardia with sine wave pattern. The patient was pulseless. He was resuscitated with 100% O_2 and DC shock (200 J) with biphasic defibrillator. Sinus rhythm was restored immediately. Simultaneously a blood sample was taken for blood gas analysis and serum electrolytes. We started amiodarone infusion to prevent recurrence of ventricular tachycardia. The blood gas analysis revealed a pH of 7.1, pCO_2 48 mm Hg (with a RR=18/min on CMV), pO_2 105 mm Hg, HCO_3^- 12mmol/L, Base Deficit/Excess (BE) -13, $S.Na^+$ 143 mmol/L and $S.K^+$ 6.09 mmol/L. Immediately treatment was started with a bolus injection 10% calcium gluconate, rapid infusion of 7.5% soda bicarbonate and glucose insulin infusion (100 ml 25% dextrose with 10 units of regular insulin) to correct hyperkalemia. After about 15 minutes the pulse rate came to near baseline values. (P=140/min. and B.P=160/96 mm Hg).

The patient was extubated and transferred to intensive care unit. Postoperative ECG was similar to preoperative ECG except for sinus tachycardia. Postoperative bedside 2D Echo was done immediately which did not reveal any abnormality. ABG after one hour was normal with S. K^+ 4.8mmol/L. The postoperative course remained uneventful and he was discharged from the ICU after 2 days.

DISCUSSION

Laparoscopic kidney transplant is a technically demanding surgery for a urologist and equally challenging for an anaesthesiologist. Extreme vigilance is paramount, owing to major perturbations in cardio-respiratory system as a consequence of steep trendelenburg position and pneumoperitoneum. Additionally, attempts to avoid deleterious effect of pneumoperitoneum on blood flow and function of transplanted kidney are required.

Anaemia, coagulopathy, hypertension and electrolyte disturbances are the most significant abnormalities of ESRD relevant to anaesthetists. Renal potassium excretion and cellular K^+ uptake play vital roles in

the body's defence against hyperkalemia [1]. These mechanisms are impaired in chronic kidney disease and such patients may show dramatic elevations in serum K^+ which can be life threatening. Though hyperkalemia is common in these patients, it is rarely seen intraoperatively; especially in a well dialysed patient [2].

Our patient underwent heparin free dialysis prior to his surgery. Preoperative blood gas analysis was normal with potassium within the normal range. The haemodynamic parameters were well controlled throughout the surgery except for the heart rate which gradually started increasing after vascular clamp release. At the conclusion of surgery, just before extubation, he suddenly developed an episode of ventricular tachycardia due to hyperkalemia. Causes of hyperkalemia during renal transplant can be transfusion of old stored blood, use of succinylcholine, muscular dystrophies, administration of potassium containing fluids or drugs, beta blockers, diabetes mellitus with decreased circulating insulin and metabolic acidosis after release of vascular clamps [3,4].

Our patient did not give any history suggestive of muscular dystrophy or weakness and neither was blood transfused nor any potassium containing fluids or drugs used intraoperatively. There have been reports where Euro-Collins solution (K^+ 115mmol/L) which is used for hypothermic flushing and preservation of kidneys prior to transplant, caused intraoperative hyperkalemia [5]. But in our case we had used HTK solution (Custodial) which has very low concentration of K^+ (KCl=9 mmol/L), thereby minimising this risk. S.K^+ increases by 0.5-0.7 mEq/L following injection of succinylcholine in both normal and uremic patients [6,7]. Preoperative S. K^+ was within normal limits in our patient, so it is unlikely that this small increase precipitated ventricular tachycardia in our patient.

Hyperkalemia in our patient occurred due to the presence of untreated acidosis (metabolic & respiratory) and lead to catastrophic arrhythmias. Acidosis leads to shift of intracellular K^+ in exchange for hydrogen ions to maintain electroneutrality [3]. The arterial blood gas analysis procured immediately after the onset of ventricular tachycardia was suggestive of acidosis (pH 7.1, pCO_2 48 mm Hg,HCO_3 12 mmol/L, BE -13). Possible causes of metabolic acidosis in our patient could be influx of lactic acid following restoration of circulation to the hypoperfused limb after completion of vascular anastomosis [3]. However, usually hyperkalemia and acidosis occur immediately on clamp release unlike in our case where it was observed almost an hour after clamp release towards the conclusion of the surgery. This could be because of the gradual escalation in S.K^+ owing to underlying respiratory acidosis in laparoscopic renal transplant, which in turn was a result of prolonged surgery in steep trendelenburg position [8]. The respiratory effects of pneumoperitoneum and trendelenburg position consist of decreased lung volumes and compliance together with the need to excrete increased CO_2 load secondary to absorption from peritoneal cavity. This leads to microatelectasis in lung bases and a ventilation–perfusion mismatch. Usually, end tidal CO_2 bears a close relationship (3-5 mm of Hg) to arterial pCO_2 ($paCO_2$) in normal patients and $EtCO_2$ monitoring has proven to be an acceptable alternative to $paCO_2$. However it may not be true in patients undergoing laparoscopic surgery in trendelenburg position because of changes in cardiac output and V/Q mismatching [9]. In our case, we had a normal $EtCO_2$ (32 mm Hg) in spite of raised $paCO_2$ (48 mm Hg) on ABG. Frequent blood sampling could have detected respiratory acidosis at an earlier stage and it could have been compensated by increasing controlled minute ventilation.

The ventricular tachycardia experienced by this patient emphasizes the importance of intraoperative prevention of acidosis and hyperkalemia.

CONCLUSION

Until definite anaesthetic guidelines for laparoscopic renal transplant are established, it would be safer to obtain frequent ABG samples intraoperatively for early detection of acidosis which could be either due to metabolic derangements owing to the underlying renal pathology or respiratory alterations as a result of increased CO_2 absorption and V/Q mismatch, especially in case of a prolonged surgery.

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